A STUDY OF PREVALENCE OF HELICOBACTER PYLORI IN DUODENAL ULCER PATIENTS

Dissertation Submitted
for the Degree of

MASTER OF SURGERY

Branch I

(GENERAL SURGERY)

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Coimbatore Medical College
Coimbatore.

CERTIFICATE

Certified that this is the bonafide dissertation done by

Dr.N.DHINAKAR BABU

and submitted in partial fulfillment of the requirement for the

Degree of **MASTER OF SURGERY**

Branch I **(GENERAL SURGERY)**

of The Tamil Nadu Dr.M.G.R. Medical University, Chennai.

DATE : **UNIT CHIEF**

DATE : **HEAD OF THE DEPARTMENT**
DEPARTMENT OF SURGERY
COIMBATORE MEDICAL COLLEGE

DATE : **DEAN**
COIMBATORE MEDICAL COLLEGE
COIMBATORE

DECLARATION

I solemnly declare that this Dissertation on **"A STUDY OF PREVALENCE OF HELICOBACTER PYLORI IN DUODENAL ULCER PATIENTS"** was done by me at Coimbatore Medical College Hospital, Coimbatore under the guidance and supervision of **Dr.S.K.PREM THAMARAI SELVI, M.S.**

Place :

Date : **DR.N.DHINAKAR BABU**

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CONTENTS

INTRODUCTION

Peptic ulcer disease is one of the most common disease affecting the general population. Although morbidity exceeds the mortality by several times, the health costs due to the disease is increasing inspite of advances in treatment and ever increasing awareness about the disease due to the high recurrence rate of the disease.

The good old adage “No acid, no ulcer” holds good even today. With excellent advances in medical control of acid secretion with proton pump inhibitors the incidence of disease has gone down. But the prevalence of the disease is ever increasing due to high recurrence rate after cessation of acid controlling therapy.

The discovery by MARSHALL and WARREN that the human gastric mucosa is colonized by a spiral gram negative organism Helicobacter pylori and its association with producing and causing recurrence of disease after adequate control of disease has opened up newer concept of conquering of a disease by mankind.

The silver lining in this that Helicobacter pylori infection can be controlled adequately with proper and adequate medical therapy has made possible for man to assert his supremacy once again.

The incidence of Helicobacter pylori is high in developing countries. There has been a reported decrease in prevalence in developed countries due to improved living conditions.

Poor socioeconomic status, poor hygiene and close personal contact are closely linked with its high prevalence.

Hence in a country like India where every penny counts, it is worth to treat the basic cause and keep the population disease free which in turn means good work force and also reduce the health expenditure on this disease.

With this background the current study is intended to study the relationship between Helicobacter pylori infection and duodenal ulcer in our hospital population.

REVIEW OF LITERATURE

HISTORICAL REVIEW

- In 1893[3] Bizzozero, an Italian pathologist reported a spiral bacteria in canine stomach.
- In 1896 similar findings were reported in other animals by Saloman[4]
- In 1906[5] the spiral bacteria were demonstrated in human stomach for the first time.
- In 1938 Doeuges demonstrated spirochaetes in stomach of necropsy specimens.
- In 1940 freedberg and Barrow used silver staining techniques to demonstrate spirochaetes in gastric mucosa.
- Further investigations by Fitzgerald and Murphy demonstrated the occurrence of gastric spiral bacteria with urease activity. This was further confirmed in gastrectomy specimens.
- In 1968 bacterial source of urease was demonstrated by absence of urease activity in germ free stomach[7].
- 1975 bacteria was associated with gastritis in 80% of resected specimen with gastric ulcer[8].

- 1983 – 1987

Robin Warren, histopathologist at Royal Perth hospital, Australia stained mucosal biopsy specimens with warthin starry stain and demonstrated the spiral bacteria[11].

Marshall treated a patient with gastritis and spiral bacterium with tetracycline and noted clearance of infection and improvement in symptoms[9].

Several attempts to culture these bacilli were unsuccessful. Finally in April 1982 when the culture plates were left unintentionally to incubate for five days colonies appeared[10].

These camphylobacter like organisms (CLO) were named camphylobacter pyloridis since they were microaerobic, curved gram negative bacteria and resembled other camphylobacteria.

- In 1987 it was changed to camphylobacteria pylori[12].
- In 1989 a new genus name was proposed and finally called as Helicobacter pylori[13]

- Helicobacter pylori was described with the following features

1. Cell motility by means of sheathed flagella
2. An external glycocalyx produced invitro in liquid media
3. Menaquinone E (Me - E) present as the major isoprenoid quinone
4. G + C content of chromosomal DNA of 35-44%

KEY NOTES IN HISTORY OF HELICOBACTER PYLORI

1893 - gastric spiral bacteria reported in animals[14]

1906 - spirochaetes demonstrated in human stomach[15]

1924 - urease activity reported in stomach[16]

1975 - Gastric spirochaetes and gastritis present in 80% of gastric ulcers[18].

1983 - CLO associated with gastritis and possibly peptic ulceration[19]

1985 - Temporal relationship between acquisition of helicobacter pylori infection and development of gastritis[20]

1987 - Eradication of helicobacter pylori leads to long term cure of duodenal ulceration[21]

1989 - Genus helicobacter formed[22]

1991 - Relationship between the organism and gastric adeno carcinoma and MALT lymphoma reported[23].

1994 - Helicobacter pylori classified as Grade I (definitive) carcinogen[24]

EPIDEMIOLOGY

- Human beings are the major reservoir of Helicobacter pylori
- Distribution of Helicobacter pylori is world wide[26]
- Most commonly the infection is acquired during childhood[27]
- The prevalence of infection increases with age from 10% at age 10 to 60% at age 60 in developed countries
- In developing countries upto 100% of children are infected by the age of 10 years[28].
- Adults rarely become infected with seroconversion rates between 0.33 – 05% per person per year[29].
- Prevalence of infection in poor socio economic status is high[28].
- Poor hygiene, close personal contact and over crowding are known risk factors for spread of Helicobacter pylori[28]
- Human milk IGA is protective for infants from Helicobacter pylori infection. This protection was demonstrable only upto 9 months.

- No difference in seroconversion rate between males and females.

- Increased helicobacter pylori seropositivity has been seen ion gastroenterologists and endoscopists[31].

- Submariners showed a higher rates of helicobacter pylori seropositivity than in other military comparison groups[32].

- Several studies found no increased risk of infection in dentists[33].

ELECTRON MICROSCOPIC PICTURE OF HELICOBACTER PYLORI

MORPHOLOGY

GENUS HELICOBACTER

SPECIES PYLORI

- It is a gram negative spiral bacillus
- It is unipolar, multiflagellate numbering around 40. The flagella of Helicobacter pylori are sheathed with a covering that is continuous with the outer membrane components of the body wall.
- It is three microns in length 0.5 to 1 milli microns in diamater.

ULTRA STRUCTURAL FEATURES

- Electron microscopy reveals the presence of 40 mm thick glycocalyx or capsule like polysaccharide rich layer outer to cell membrane.

PHYSIOLOGICAL PROPERTIES

- Helicobacter pylori is microaerophile growing best in an atmosphere of 5% oxygen with 5-10% CO_2 on blood containing media.

Eg. Oxoid brain heart infusion agar

5% blood agar enriched with 1% isovitale x.

Enriched chocolate agar broth.

It appears as translucent, circular, convex, colonies of about 2 millimeter in diameter.

BIOCHEMICAL CHARACTERISTICS

- Helicobacter pylori produces
- Catalase
- Cytochrome oxidase
- Protease
- Phospholipase A_2 C
- Acid phosphatase
- Alkaline phosphatase
- Leonine arylamidase
- Naphthol AS-B_1 phosphohydrolase
- Esterases C_4, C_8
- Gammaglutamyl transpeptidase

It produces urease which is present in the cytoplasm and surface of the organism.

Urease produces the following action

$$\text{Urea} + H^+ + \text{water} \xrightarrow{\text{urease}} \text{bicarbonate} + \text{ammonium}$$

$$C = O(NH_2)_2 + H^+ + 2H_2O \xrightarrow{\text{urease}} HCo_3 + 2(NH_4)$$

Ammonium formed creates an alkaline environment around the organism which protects the organism from the gastric acid assault enhancing survival.

GENOMIC DNA

It is a single circular molecule with a mean size of 1.71 Mb ranging from 1.40 – 1.73 Mb and base composition in the range 35 – 37 mol % G + C.

FATTY ACID COMPOSITION

The major cellular fatty acids are tetradeconoic acid and is 11, 12 methylene octadecanoic acid with smaller amounts of hexadeconoic acid and 3 hydroxy decanoic acid.

The main respiratory quinone is menaquinone - 6 (MK - 6)

Extrachromosomal DNA plasmid DNA is present in 45% of strains.

The lipopolysaccharide part of Helicobacter pylori express lewis x and y blood group antigens in 80% strains which are not found in other gram negative organisms.

HABITAT

The natural habitat of helicobacter pylori is human stomach.

Any part of the stomach may become colonized but the mucus secreting epithelium of the antrum is the favoured site.

Colonization of areas of gastric metaplasia or ectopic gastric mucosa in other parts of the gastrointestinal track.

Helicobacter pylori has been detected in dental plaque by cultured.

It has also been cultured from saliva of a patient with gastritis.

Survival in gastric mucosa

It lives beneath the mucus layer that covers the gastric mucosa.

It lies deep inside the crypts of gastric glands.

Spiral shape and motility makes it able to resist peristalsis.

Urease produces a protective alkaline environment around the organisms. This buffers the acid assault.

Microaerophilic nature is suited for the environment and adhesions help in permanent residency.

Protease produced by helicobacter pylori helps to establish itself in this stomach wall bypassing localized inflammation.

MODE OF TRANSMISSION

Potential mode of transmission is by three ways

1. FAECO ORAL TRANSMISSION

Water has been a source of Helicobacter pylori infection. The organism has been isolated from faeces[34]. Polymerase chain reaction assays have demonstrated the presence of organism in food and drinking water.

2. ORAL - ORAL TRANSMISSION

Helicobacter pylori has been isolated from the oral cavity. There is evidence of transmission between spouses although it could be due to common source infection[35]. It is also possible that re- infection may occur by person to person transmission between spouses[36].

3. GASTRO ORAL TRANSMISSION

In children it may be due to reflux and vomiting. A physician become infected with Helicobacter pylori after he gave mouth to mouth resuscitation to a patients with positive Helicobacter pylori status who had recently vomited[37].

Another important source is iatrogenic transmission in individuals who have undergone endoscopy procedures with a contaminated pH electrode or biopsy forceps.

PATHOGENESIS

Helicobacter pylori is the main cause of duodenal and gastric ulceration. It is also a major risk factor for adenocarcinoma and lympoma.

Development of the disease is influenced by

1. Virulence of the infecting strain
2. genetic susceptibility of the host
3. Environmental co-factors

1. VIRULENCE FACTORS

A host of factors contribute to the ability of Helicobacter pylori to colonize gastric mucosa, evade host defences and damage host tissue.

Helicobacter pylori localizes on epithelial surface beneath the mucous layer.

It attaches to epithelial cells by adhesin.

It can also invade the epithelial cells,

It produces a cytotoxin which is vacuolising.

It contains cytotoxin associated gene A ("Cag" segment of DNA).

Damage is caused by

- reduced epithelial cell turnover
- reduced surface hydrophobicity
- increased lipolysis
- release of chemotactic factors
- activation of classical complement pathway
- stimulation of G cells

Other factors include

Adhesins, protease, lipase, catalase, superoxide dismutase and platelet activating factor.

Host factors

- Host HLA type[39]
- Blood group antigen type[40]

Environmental factors[30]

- Living conditions particularly in childhood since most of the infections are acquired in childhood.
- Smoking
- Dietary factors

Gastric colonization is a pre requisite for Helicobacter pylori associated disease. For this both urease and motility are vital.

Infection is lifelong unless eradication treatment is given. This indicates that Helicobacter pylori evades host immune defences effectively.

Gastric inflammation in Helicobacter pylori infected individuals represents the host immune response to the organism[43].

Gastritis caused by Helicobacter pylori is characterized by surface epithelial degeneration, infiltration of mucosa by chronic inflammatory cells (lymphocytes, plasma cells and occasional eosinophils) and neutrophils.

The effect of gastritis on gastric acid secretion depends upon to what extent it involves the antrum or body mucosa.

Gastritis confined to antrum without atrophy results in hypersecretion of acid. This is due to stimulated increased release of gastrin[42]. This is the pattern of gastritis seen in individuals with duodenal ulceration[41].

Now it is proved that increased gastric release is due to deficient somatostatin.

Helicobacter pylori infected patients have significantly higher basal. 24 hours meal stimulated and gastrin releasing peptide stimulated gastrin levels than normal individuals

Basal and gastrin releasing peptide stimulated acid secretion are increased only in symptomatic individuals as compared to asymptomatic infected patients.

When the gastritis involves the mucosa of body then parietal cells are impaired by the inflammatory process. There is no increase in acid secretion. This will lead to hypochlorhydria or achlorhydria.

Two types of metaplasia can occur in chronic gastritis

1. Pyloric metaplasia

There is replacement of the fundic type glands by mucus secreting glands.

2. Intestinal metaplasia

Progressive replacement of gastric mucosa by intestinal type epithelium of either small or large bowel type including goblet cells, absorptive cells (brush border), paneth cells and other endocrine cells[44].

CLINICAL ASSOCIATIONS

Helicobacter pylori has been implicated in the following disease[26]

1. Duodenal ulcer
2. Gastric ulcer
3. Gastroduodenitis
4. Carcinoma body and antrum
5. MALT asso gastric lymphoma

Extra intestinal manifestations

1. Ischaemic heart disease
2. Cerebro vascular accident
3. Raynaud's phenomenon
4. Idiopathic migraine
5. Henoch schonlein purpura
6. Sjogren's syndrome
7. Autoimmune thrombocytopenia
8. Extragastric MALT lymphomas
9. Siderophenic anaemia
10. Idiopathic chronic urticaria

11. Acne rosaceae
12. Alopecia areata
13. Cirrhosis
14. Cholesterol stones
15. Chronic cholecystitis
16. Bronchictasis
17. Sudden infant death syndrome
18. Non ulcer dyspepsia

- It is also present in asymptomatic normal gastric mucosa.
- 95% of patients with duodenal ulcers and 80% of patient with gastric ulcer are infected with the bacterium and its eradication greatly diminishes the recurrence rate.
- Evidence in support of Helicobacter pylori as a causative agent in peptic ulcer disease

1. Follow up of the natural history of the subjects infected with Helicobacter pylori revealed 11% developed peptic ulcers compared with less than 1% of subjects without gastritis[45].

2. World wide consistent association of Helicobacter pylori and ulcer disease[46].

3. Multiple prospective treatment trials indicate that the eradication of Helicobacter pylori prevents recurrent duodenal ulcer in most cases[47].

4. The effects of the infection were illustrated after deliberate ingestion of a suspension of the organism (Koch's postulate)[20].

DIAGNOSTIC METHODS

These can be divided into

1. those requiring endoscopy
2. those not requiring endoscopy

These can also be divided into those done for initial diagnosis (pre treatment) and for assessment of cure (post treatment).

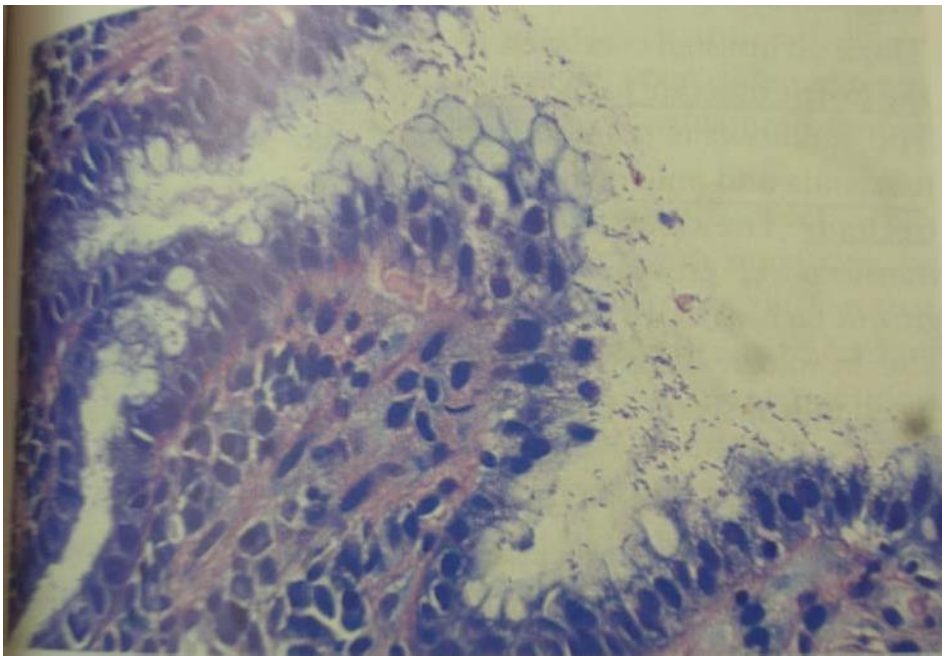
Method involving endoscopy

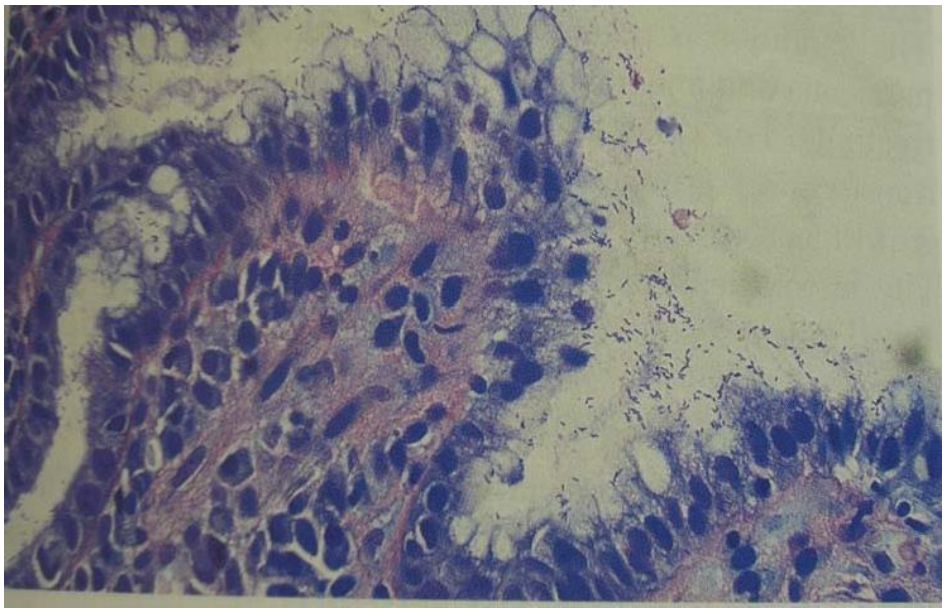
The critical role of endoscopy in the diagnosis of helicobacter pylori is to obtain gastric mucosal biopsies which are utilized for both direct and indirect methods of detecting the presence of organism.

1. Direct method are
    a. Culture and
    b. Histology

2. Indirect methods
    a. Urease testing

GIEMSA STAINING OF HELICOBACTER PYLORI





They can also be divided into

Invasive	**Noninvasive**
1. Urease	1. Serology
2. Culture	2. Urea breath test
3. Histology	
4. PCR	

1. CULTURE

This is not routinely done.

Sensitivity achieved is more than 95% in some series. It takes 2-5 days time. Antibiotic sensitivity can be determined.

It must be incubated at 37^0C under high humidity for a maximum of 24 - 72 hours.

2. HISTOLOGY

This is the Gold Standard for the diagnosis of Helicobacter pylori on gastric biopsies.

Needs reasonably large bits for study.

Prepyloric antrum has been the preferred site of biopsy.

Staining methods in use are

1. GIEMSA
2. Eosin and haematoxylin
3. Warthin starry silver stain

3. RAPID UREASE TEST (RUT)

Developed by Marshall. CLO test is the first commercially available test.

Helicobacter pylori urease hydrolysis the urea contained in the agar gel of the test solution to produce ammonia. This causes a pH rise and change of colour from yellow to pink.

Phenol red is used as indicator.

The test is interpreted upto 24 hrs after inoculating the biopsy specimen.

The overall sensitivity of the test was 90%[48].

The other commercially available RUT are

1. Hp test
2. Pyloritek - strip test

4. POLYMERASE CHAIN REACTION

It is currently done for research purposes to identify different strains.

5. ANTIBODY DETECTION

Helicobacter pylori IgG antibodies are detected by

1. ELISA
2. Latex agglutination test

They are non-invasive and inexpensive tests

It is a reasonable choice for initial diagnosis of helicobacter pylori infection.

6. UREA BREATH TEST

^{13}C and ^{14}C urea breath test measures the activity of Helicobacter pylori urease.

Oral radioactive carbon labeled urea is given. If Helicobacter pylori is present in the stomach, urease hydrolyses the labeled urea releasing labeled bicarbonate that is transported in the blood to the lungs and enhaled as carbondi-oxide.

The breath is collected and radioactivity level is measured. This test is done in children.

This test should not be used in patients who have taken proton pump inhibitors, bismuth compounds or antibiotics.

Other assays

1. Detection of antibodies in urine.
2. measurement of labelled serum bicarbonate following administration of radioactive labeled carbon.
3. Measurement of radioactivity in urine.
4. Measurement of ^{15}N labeled ammonia which is absorbed and excreted in the urine.

POST TREATMENT TEST

INDICATIONS

1. Patients with bleeding duodenal ulcer on completion of eradication therapy.
2. Patients with recurrent peptic ulcer symptoms.

Routine testing is not advocated in patients with uncomplicated duodenal ulcer.

This test should be performed ideally four weeks after completion of eradication therapy.

TREATMENT

The aim of treatment is to eradicate Helicobacter pylori. Eradication is defined as negative tests for Helicobacter pylori atleast 28 days after the end of antimicrobial therapy.

Ideal therapy for Helicobacter pylori should be

- Simple
- Safe
- free from side effects
- efficacy is 100%
- low cost
- acceptable to the patient
- available to the patient

Therapies available

1. Dual therapy
2. Triple therapy
3. Low dose triple therapy
4. Quadruple therapy

DUAL THERAPY

Table No. 1

Drug	Dose	Duration	Eradication
Omeprazole Amoxycillin	20 - 40 mg BD 750 mg TDS	2 wks	50 – 85 %

Table No. 2

Drug	Dose	Duration	Eradication
Ranitidine bismuth citrate Amoxycillin	400 - 500 mg BD 500 mg QID	2 wks	65 %

Table No. 3

Drug	Dose	Duration	Eradication
Omeprazole Clarithromycin	40 mg OD 500 mg TID	2 wks	80 %[49]

Table No. 4

Drug	Dose	Duration	Eradication
Ranitidine bismuth citrate Clarithromycin	400 mg BD 500 mg BD	2 wks	80%[50]

TRIPLE THERAPY

Table No. 5

Drug	Dose	Duration	Eradication
Omeprazole	40 mg OD	7 days	95 %
Amoxycillin	500 mg TID		
Metronidazole	400 mg TID		

Table No. 6

Drug	Dose	Duration	Eradication
Ranitidine	300 mg OD	12 days	90 %
Amoxycillin	750 mg TID		
Metronidazole	500 mg TID		

Table - 7

LOW DOSE TRIPLE THERAPY

Drug	Dose	Duration	Eradication
PPI Clarithromycin Metronidazole	20 mg OD or BD 250 mg BD 400 mg BD	7 days	90 %

Table - 8

LOW DOSE TRIPLE THERAPY

Drug	Dose	Duration	Eradication
PPI Clarithromycin Metronidazole	20 mg BD 1 gm BD 250 - 500 mg BD	7 days	90 %

Table - 9

QUADRUPLE THERAPY

Drug	Dose	Duration	Eradication
PPI	20 mg OD or BD	7 days	85 - 95 %
Colloidal bismuth citrate	120 mg QID		
Tetracycline	500 mg QID		
Metronidazole	400 mg TID		

Low dose triple therapy is the most preferred therapeutic regimen. It results in a high eradication rate and most economical regimen[51].

Clarithromycin based drug regimens achieved a good eradication rate with a definitive advantage is smokers[54].

Quadruple drug regimen using bismuth with two antibiotics and proton pump inhibitors does not seen to offer any additional advantages[55].

AIM OF STUDY

A random group of adult male and female patients who had epigastric pain were group to formulate this study aiming at

1. To find out the incidence of Helicobacter pylori in proven cases of duodenal ulcer.

2. To find out the association of Helicobacter pylori in individuals with normal upper gastrointestinal tract.

3. To analyse the incidence of Helicobacter pylori with reference to
    1. Age
    2. Sex
    3. Socio economic status
    4. Symptoms
    5. Signs

4. To compare with other studies

MATERIALS AND METHODS

DESIGN OF STUDY

This is a case control study in detecting the prevalence of Helicobacter pylori among 50 patients with duodenal ulcer and another 50 patients with normal upper gastrointestinal tract as controls.

This study involves randomized controlled trials in which both the study group and control group were selected according to set selection criteria as detailed below.

STUDY PERIOD

The study was conducted from May 2005 to March 2006 in patients from around Coimbatore who were subjected to upper gastrointestinal endoscopy.

STUDY CENTRE

This study was carried out at Department of Surgery and Department of gastroenterology of Coimbatore Medical College Hospital, Coimbatore.

ENDOSCOPE AND BIOPSY FORCEPS

SUBJECT SELECTION

Totally 100 patients were selected from those who had undergone upper gastro intestinal endoscopic evaluation.

50 patients with duodenal ulcer were grouped as cases.

50 patients with normal upper gastro intestinal endoscopy were grouped as controls.

SELECTION CRITERIA

- Endoscopically proved duodenal ulcer was made as an absolute pre requisite.
- Patients above 12 yrs of age
- No other obvious findings like gastric ulcer, baretts esophagus, oesophagitis in the endoscopy.
- No history of previous NSAID ingestion

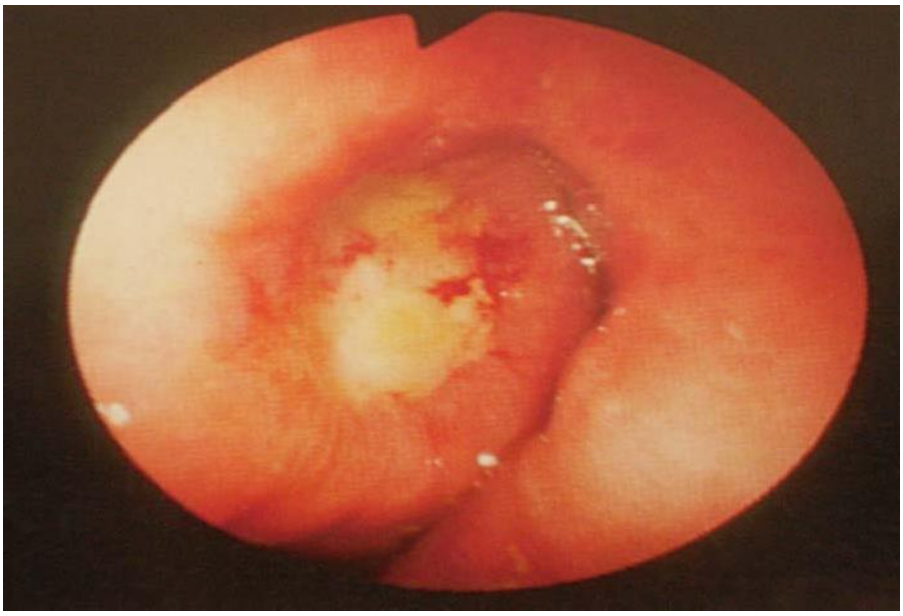
TECHNIQUE

Upper gastrointestinoscopy was performed under local anaesthesia after fully explaining the procedure to the patient.

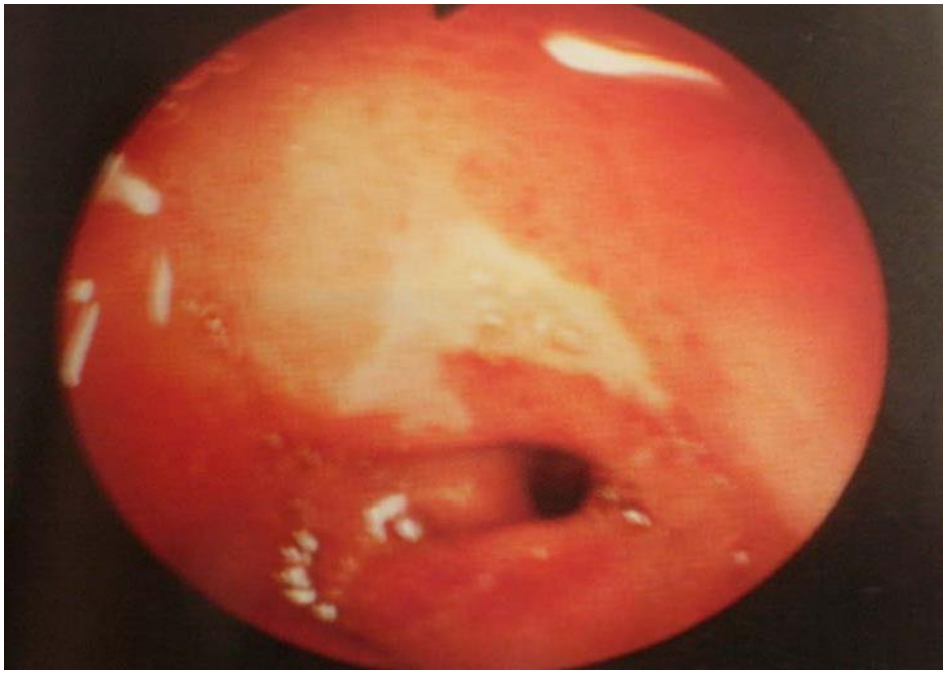
Patients were asked to lie in left lateral position with both hips and knee flexed and arms between the legs.

NORMAL DUODENAL MUCOSA

DUODENAL ULCER



DUODENAL ULCER WITH PYLORIC STENOSIS



DUODENAL ULCER WITH INFLAMMATORY CHANGES

A plastic mouth gag was inserted and held firmly by the assistant.

Local anaesthetic spray was used before inserting the endoscope. Endoscope was passed into the oropharynx crossing the cricopharynx into the oesophagus all the way encouraging the patient to swallow.

Once the tube has crossed the cricopharyngeal sphincter tube can be easily passed without the patients aid.

The oesophagus stomach, first and second part of the duodenum were visualized and screened for any pathology.

If the patient was found to have a duodenal ulcer biopsy was taken from multiple sites in the antrum of stomach.

Endoscope and biopsy forceps were disinfected.

RAPID UREASE TEST

Urea (10%) 10 gm

Distilled water 100 ml

UREASE SOLUTION WITH BIOPSY BIT INOCULATED AND PHENOL RED ADDED

UREASE TEST POSITIVE

Monobasic potassium dihydrogen phosphate 0.005 %

Phenol red indicator

Dissolved in distilled water

Autoclave if for 30 minutes

PROCEDURE

1 ml liquid urease medium is dispensed in autoclaved bottles. Biopsy bits are inoculated in the endoscopy room and one drop of phenol red is added and observed for 30 mins to 1 hours. If there is colour change from yellow to pink it is considered positive.

Fresh solutions are prepared every week.

RESULTS

The full collected details of cases and controls have been recorded in the master chart.

This study is composed of 100 patients.

- 50 patients with duodenal ulcer
- 50 patients with normal gastrointestinal tract

Age group included in the duodenal ulcer group and control group is between 15 – 80 yrs.

Sex distribution in the study is as follows

In case out of 50 patients 35 were male and 15 were female

Out of 50 controls 20 were male and 30 were female

The following results and observations were arrived at after analyzing the collected data from this study.

Helicobacter pylori in duodenal ulcer and controls

The prevalence of helicobacter pylori is high among duodenal ulcer patients (78%) when compared with controls (26%).

The ratio of helicobacter pylori positive versus negative incidence in duodenal ulcer patients is 4 : 1. (Chart - 1).

The ratio of helicobacter pylori positive versus negative incidence in controls is 1:3. (Chart - 2).

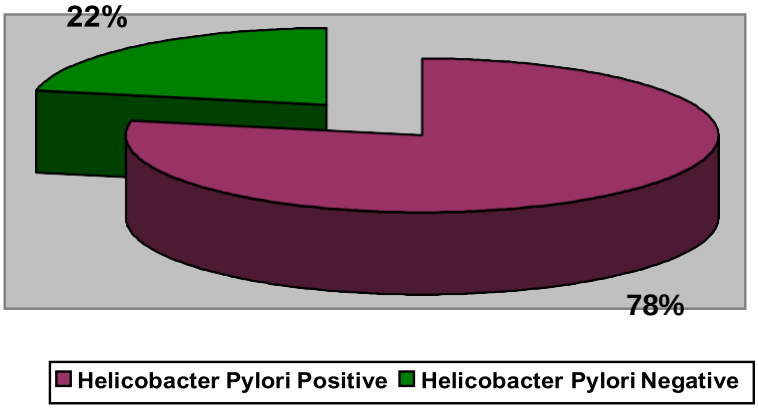
PREVALENCE OF HELICOBACTER PYLORI

Table No. 10

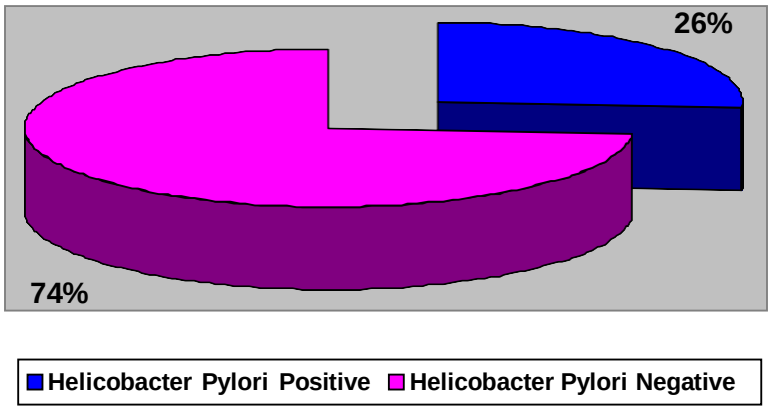
Study Group	Nos	Helicobacter pylori +ve		Helicobacter pylori -ve	
		Nos	%	Nos	%
Duodenal ulcer	50	39	78%	11	22%
Control	50	13	26%	37	74%

The incidence of Helicobacter pylori in both duodenal ulcer patients and control group increases progressively from younger age group to older age group.

PREVALENCE OF HELICOBACTER PYLORI



PREVALENCE OF HELICOBACTER PYLORI



AGE GROUP DISTRIBUTION CHART FOR CASES

Table No. 11

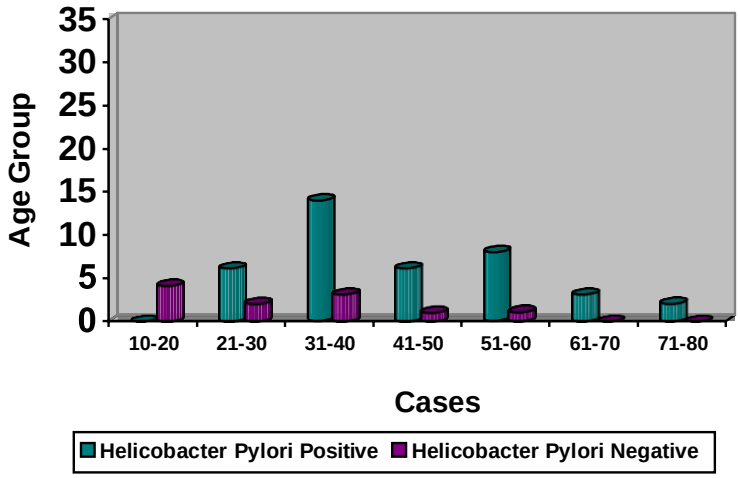
S. No.	Age (yrs)	No. of cases	H.P. +ve	H.P. -ve	%	H.P. +ve	H.P. -ve	M	F
1	10-20	4	-	4	8	0	100	3	1
2	21-30	8	6	2	16	75	25	5	3
3	31-40	17	14	3	34	82	18	10	7
4	41-50	7	6	1	14	86	14	6	1
5	51-60	9	8	1	18	89	11	8	1
6	61-70	3	3	-	6	100	0	2	1
7	71-80	2	2	-	4	100	0	1	1

AGE GROUP DISTRIBUTION CHART FOR CONTROLS

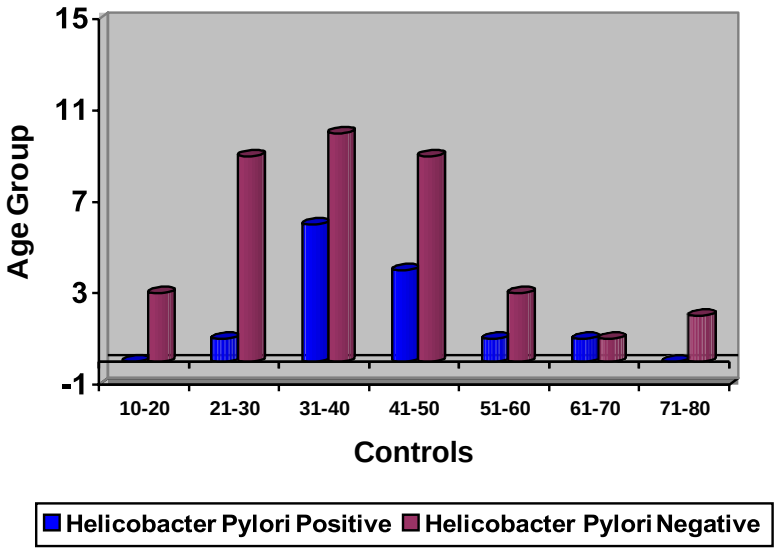
Table No. 12

S. No.	Age (yrs)	No. of cases	H.P. +ve	H.P. -ve	%	H.P. +ve	H.P. -ve	M	F
1	10-20	3	0	3	6	0	100	1	2
2	21-30	10	1	9	20	10	90	4	6
3	31-40	16	6	10	32	37.5	62.5	8	8
4	41-50	13	4	9	26	31	69	4	9
5	51-60	4	1	3	8	25	75	0	4
6	61-70	2	1	1	4	50	50	1	1
7	71-80	2	0	2	4	0	100	2	0

AGE GROUP DISTRIBUTION OF CHART FOR CASES



AGE GROUP DISTRIBUTION OF CHART FOR CONTROLS



There is a male predominance of Helicobacter pylori prevalence when compared to females in control group who have normal upper gastrointestinal tract (35% Vs 20%).

But there is no similar sex predilection in the prevalence of Helicobacter pylori in duodenal ulcer patients (77% Vs 80%).

Table No. 13

HELICOBACTER PYLORI AND SEX INCIDENCE

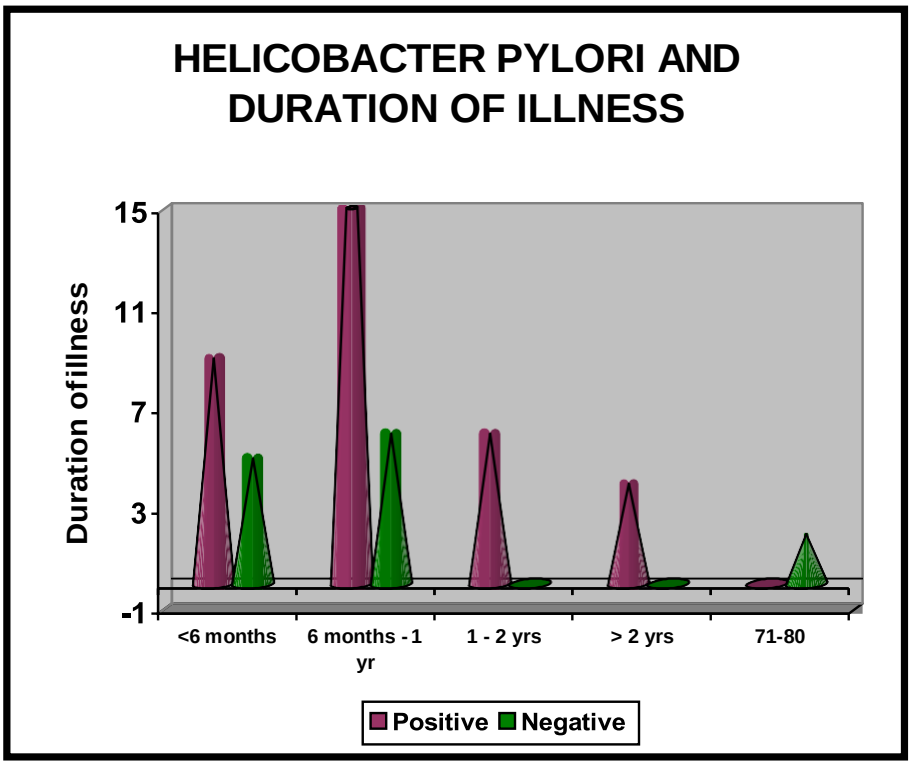
Study Group	Helicobacter pylori Positive				Helicobacter pylori Negative			
	M	**%**	**F**	**%**	**M**	**%**	**F**	**%**
Duodenal ulcer	27	77	12	80	8	23	3	20
Control	7	35	6	20	13	65	24	80

HELICOBACTER PYLORI DURATION OF ILLNESS

The prevalence of Helicobacter pylori increased with chronicity of symptoms and duration of illness.

Table No. 14

Duration of Illness	No. of Patients	Helicobacter pylori		Positive Percentage
		Positive	Negative	
< 6 months	14	9	5	64
6 months - 1 yr	26	20	6	77
1 - 2 yr	6	6	0	100
> 2 yr	4	4	0	100


HELICOBACTER PYLORI AND DURATION OF ILLNESS
Duration ofillness
15
11
7
3
-1
<6 months
6 months - 1 yr
1 - 2 yrs
> 2 yrs
71-80
Positive
Negative

DISCUSSION
AND COMPARATIVE ANALYSIS

Helicobacter pylori and non steroidal anti-inflammatory drugs have been considered as two major etiological agents in peptic ulcer disease and acid remains as a contribution than a prime cause[56].

The maxim “once an ulcer, always an ulcer” is no longer appropriate after the discovery of Helicobacter pylori.

This study is intended to find out the relation between Helicobacter pylori and duodenal ulcer and to correlate and compare with other studies.

This study shows that the prevalence of Helicobacter pylori in duodenal ulcer patients is 78% which is correlated with other studies below.

The prevalence of Helicobacter pylori in normal gastrointestinal tract is 26%. In most of the western studies the prevalence is ranging between 0-255.

Indian study by Prabhu et al showed a prevalence of 23.5%.

This present study has shown high prevalence of Helicobacter pylori in duodenal ulcer patients (78%) than in normal upper gastrointestinal tract mucosa (26%). There is a definite relationship between Helicobacter pylori and duodenal ulcer.

Table No.15

Name of study	Year of study	Patients with duodenal ulcer	
		No. of Patients	Helicobacter pylori positive %
Marshall and Warren	1984	13	100
Langenberg et al	1984	9	100
Burnett et al	1984	7	57
Lambert et al	1985	61	95
Mc Nulty and Watson	1986	20	95
Booth et al	1986	25	78
Von Wulffen et al	1986	54	83
Pettross et al	1986	25	60
Humphries et al	1986	64	93
Lamouliatte et al	1986	21	90
Fiocca et al	1987	30	88
Hirchl et al	1987	53	98
Graham et al	1987	85	91
Rauws and Tytgat[57]	1989	165	100
Present Study	2006	50	78

Table No. 16

Name of study	Year of study	Patients with duodenal ulcer	
		No. of Patients	Helicobacter pylori positive %
Forrest et al	1984	25	24
Jones et al	1984	15	0
Langenberg et al	1984	19	5
Meyrick Thomal et al	1984	9	0
Rollason et al	1984	10	10
Lambert et al	1985	21	5
Buck et al	1986	7	14
Jiang et al	1987	15	20
Raskov et al	1987	50	20
Johnson et al	1988	105	10
Prabhu et al (Indian)	1993	17	23.5
Present Study	2006	50	26

The prevalence rate of helicobacter pylori increases progressively with age[62]. Several studies show that it is 10-20 in young adults, 50% between 50-60 yrs and increases to 75% over 65 yrs of age[62].

Our study has well correlated with the above fact that it shows increasing prevalence with age in patients with duodenal ulcer.

In the present study the prevalence of helicobacter pylori infection is almost double in males when compared to females in those with normal upper gastrointestinal tract.

This correlates with the fact that helicobacter pylori infection is a male preponderance disease. Certain studies are available to support this. A study conducted in rural Columbian andes communities found higher incidence of infection rates in young males[59]. A birth cohort of 21 year olds in Newzealand revealed a higher seropositivity in males[60]. Males in a variety of ethnic groups in California had higher seropositivity than females[61].

In duodenal ulcer patients the prevalence is almost equal in both male and female.

The prevalence of helicobacter pylori increased with chronicity of the disease.

But this cannot be considered very significant because the patients above 1-2 yrs duration is a very small group when compared with patients who have lesser duration disease. It may require a bigger sample and equal number of patient in both groups to compare.

The various symptoms were analysed in relation to prevalence of helicobacter pylori.

The most common and prominent symptom is abdominal pain and periodicity.

Abdominal pain is present in almost all patients.

Periodicity is present in 87% of cases in whom the rapid urease test was +ve when compared to 72% in whom rapid urease test was -ve.

The next common symptom was nocturnal pain. It was present in 64% of patients with helicobacter pylori positive when compared to 55% of patients with helicobacter pylori negative.

Other symptoms include G.I. bleed and retrosternal burning sensation whose prevalence was 28% and 87% respectively. Water brash was present in 38%.

Table No. 17

Symptoms	Helicobacter pylori +ve (39)		Helicobacter pylori -ve (11)	
	No. of Patients	Percentage	No. of Patients	Percentage
Periodicity	34	87	8	72
Nocturnal pain	25	64	6	55
Retrostrenal burning pain	34	87	9	82
Water brash	15	38	3	27
GI bleeding	11	28	0	0

A study conducted at AIIMS - New Delhi in 1980 the nocturnal pain was present only in 40% of patients and less than 10% had gastrointestinal bleeding. But recently the prevalence of nocturnal pain seems to be increasing[64].

This may be because of change in lifestyle with longer working hours and increased stress.

Although complications are not increased by presence of helicobacter pylori is increased in patients developing complications. This fact can be used for therapeutic purpose that eradication may reduce the further complications.

Eradication of helicobacter pylori in uncomplicated ulcers results in higher eradication rates and lower recurrence rate less than 10%. This results in lesser complication rate.

A combination of rapid urease test and brush cytology was found to be the most sensitive and least expensive test.

CONCLUSION

To conclude this study conducted at Department of Surgery, Coimbatore Medical College which comprise of 100 patients of whom 50 has duodenal ulcer and another 50 with normal upper gastrointestinal tract to under went upper gastrointestinal tract endoscopy the following facts are highlighted.

1. This study reveals that rapid urease test is very easy, less expensive out patients procedure to diagnose helicobacter pylori infection.

2. There is a definitive relationship between helicobacter pylori and duodenal ulcer.

3. It appears to play a causative role in the pathogenesis of duodenal ulcer.

4. There is a 78% prevalence of helicobacter pylori in duodenal ulcer patients when compared to 26% prevalence in those with normal Gastrointestinal tract.

5. It also correlated with other studies in the prevalence of helicobacter pylori in duodenal ulcer.

6. It reveals that the prevalence of helicobacter pylori is doubled in males when compared to females in controls.

7. But the prevalence is equal in both sexes in duodenal ulcer patients.

8. It reveals that there is a positive relationship between helicobacter pylori and duration of disease.

9. It reveals that helicobacter pylori infection increase progressively with age.

10. Helicobacter pylori prevalence in duodenal ulcer patients can be detected by an outpatient procedure (endoscopy) with aid of reliable, cheap rapid urease test. On detection eradication can be done effectively with available drug combinations. This results in prevention of complications in duodenal ulcer patients and also high cure rate.

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Η.ΠΥΛΟΡΙ ΔΥΟΔΕΝΑΛ ΥΛΧΛΕΡ

ΠΡΟΦΟΡΜΑ

NAME : AGE : SEX : I.P. NO :

WARD : UNIT : D.O.A.: D.O.D. :

HISTORY	YES	NO
NSAIDS INGESTION		
ABD. PAIN		

DURATION	YY	MONTH	DAYS

NOCTURNAL RELATIONSHIP			
PERIODICITY			

	INCREASED/DECREASED		NOCHANGE
RELATED TO FOOD			
RELATED TO ANTACIDS			
RELATED TO VOMITING			

	YES	NO
WATER BRASH	☐	☐
ACID ERUCTATION	☐	☐
HEART BURN	☐	☐
G.I.BLEEDING	☐	☐

ENDOSCOPY

OESOPHAGUS	NORMAL	NOT
STOMACH	NORMAL	NOT
DUODENUM	NORMAL	NOT
GASTIC OUTLET OBSTRUCTION	PRESENT	NOT
ULCER BLEEDING	PRESENT	NOT
IMPRESSION	DU	NORMAL
RAPID UREASE TEST	POSITIVE	NEGATIVE

MASTER CHART

CASES

Sl. No.	Name	Age /Sex	IP No.	Pain	Duration	Periodicity	Noct. Pain	Related to			Water brash	Heartburn & Acid Eructation	G.I. Bleed	Goo	Endoscopy	RUT
								Food	Antacid	Vomiting						
1	Raman	28 / M	1408/05	+	4 mon	+	+	-	-	-	-	-	-	-	DU	-
2	Backiam	48 / F	1458/05	+	2 mon	-	+	+	-	-	+	+	-	-	DU	+
3	Senthilkumar	18 / M	1518/05	+	6 mon	-	-	-	-	-	-	-	-	-	DU	-
4	Siva	31 / M	1526/05	+	3 mon	+	+	+	-	-	+	+	+	-	DU	+
5	Ramalingam	44 / M	1534/05	+	11mon	+	+	+	-	+	+	+	-	-	DU	+
6	Xavier	26 / M	1603/05	+	7 mon	+	+	-	-	-	-	+	-	-	DU	+
7	Bama	33 / F	1679/05	+	2 mon	+	+	-	-	-	+	+	+	-	DU	+
8	Kandasamy	53 / M	1681/05	+	10mon	+	-	-	-	-	+	+	-	-	DU	+
9	Michael	34 / M	1712/05	+	11mon	+	+	-	-	-	-	-	-	-	DU	+
10	Ahmed	73 / M	1741/05	+	7 mon	+	+	-	-	-	-	+	-	-	DU	+
11	Sooman	35 / F	1750/05	+	4 mon	+	+	-	-	-	-	+	+	-	DU	+
12	Ramasamy	63 / M	1790/05	+	5 mon	+	+	-	-	-	-	+	-	-	DU	+

Sl. No.	Name	Age /Sex	IP No.	Pain	Duration	Periodicity	Noct. Pain	Related to			Water brash	Heartburn & Acid Eructation	G.I. Bleed	Goo	Endoscopy	RUT
								Food	Antacid	Vomiting						
13	Saravanan	16 / M	1796/05	+	11mon	-	+	+	-	-	-	-	-	-	DU	-
14	Kannan	21 / M	1810/05	+	8 mon	-	+	-	-	+	+	+	+	-	DU	+
15	Sundaralingam	56 / M	1815/05	+	7 mon	+	+	-	-	-	+	+	-	-	DU	+
16	Kanaga	38 / F	1824/05	+	11mon	+	+	-	-	-	-	+	-	-	DU	+
17	Ramasamy	41 / M	1844/05	+	7 mon	+	+	-	-	-	-	-	-	-	DU	+
18	Ramayee	74 / F	1902/05	+	10mon	+	-	-	-	-	-	-	-	-	DU	+
19	Sundaram	36 / M	1905/05	+	5 mon	-	-	-	-	-	+			-	DU	-
20	Kumaravel	32 / M	1896/05	+	8 mon	+	+	+	-	+	-	+	+	+	DU	+
21	Lakshmi	55 / F	1910/05	+	3 yrs	+	+	-	-	+	-	+	-	-	DU	+
22	Jeeva	16 / F	1912/05	+	10mon	+	+	-	-	-	-	-	-	-	DU	-
23	Thangam	34 / F	2004/05	+	1½ yrs	+	-	-	-	-	-	+	+	-	DU	+
24	Durai	58 / M	2110/05	+	1½ yrs	+	-	+	-	+	-	+	-	+	DU	+
25	Karuppasami	38 / M	2118/05	+	4 yrs	-	-	-	-	+	+	+	-	-	DU	+
26	Mani	29 / M	2306/05	+	7 mon	+	+	-	-	-	-	+	-	-	DU	+
27	Mahalingam	53 / M	2812/05	+	10mon	+	+	+	-	-	-	+	-	-	DU	+
28	Fathima	26 / F	2348/05	+	8 mon	+	-	-	-	-	+	+	-	-	DU	-
29	Mangalam	36 / F	2566/06	+	2 yrs	+	-	-	-	-	-	-	-	-	DU	+

Sl. No.	Name	Age /Sex	IP No.	Pain	Duration	Periodicity	Noct. Pain	Related to			Water brash	Heartburn & Acid Eructation	G.I. Bleed	Goo	Endoscopy	RUT
								Food	Antacid	Vomiting						
30	Mani	33 / M	2680/05	+	11mon	+	-	-	-	-	-	+	-	-	DU	+
31	Kanagavel	24 / M	2730/05	+	8 mon	+	+	-	-	-	+	+	-	-	DU	+
32	Sundaram	38 / M	2840/05	+	5 yrs	+	+	-	-	-	-	+	+	+	DU	+
33	Sumathi	38 / F	2936/05	+	7 mon	+	+	+	-	-	-	-	-	-	DU	-
34	Lakshmanan	61 / M	2954/05	+	2 yrs	+	-	-	-	-	-	+	-	-	DU	+
35	Vairavan	56 / M	3060/05	+	10mon	+	-	-	-	+	-	+	-	-	DU	+
36	Suganthi	32 / F	3079/05	+	3 mon	-	-	-	-	-	+	+	-	-	DU	+
37	Subbammal	65 / F	90/06	+	1½ yrs	+	-	-	-	-	-	+	+	-	DU	+
38	Suresh	18 / M	125/06	+	3 mon	-	-	-	-	-	-	+	-	-	DU	-
39	Kanagavel	45 / M	250/06	+	5 mon	+	-	-	-	-	+	+	-	-	DU	+
40	Swaminathan	39 / M	270/06	+	2 mon	+	-	-	-	-	-	-	-	-	DU	-
41	Gopal	48 / M	282/06	+	9 mon	+	-	-	-	-	+	+	-	-	DU	-
42	Ramasamy	32 / M	321/06	+	4 mon	-	+	-	-	-	-	+	+	-	DU	+
43	Maran	55 / M	340/06	+	2 yrs	+	+	+	-	-	+	+	-	+	DU	+
44	Latha	28 / F	359/06	+	8 mon	+	+	-	-	-	-	+	-	-	DU	+
45	Krishnan	49 / M	393/06	+	4 yrs	+	-	-	-	+	-	+	+	+	DU	+
46	Xavier	37 / M	420/06	+	9 mon	+	+	-	-	-	-	+	-	-	DU	+

Sl. No.	Name	Age /Sex	IP No.	Pain	Duration	Periodicity	Noct. Pain	Related to			Water brash	Heartburn & Acid Eructation	G.I. Bleed	Goo	Endoscopy	RUT
								Food	Antacid	Vomiting						
47	Kannan	56 / M	448/06	+	7 mon	+	-	-	-	-	-	+	+	-	DU	+
48	Rukmani	30 / F	460/06	+	3 mon	+	+	-	-	-	-	+	-	-	DU	+
49	John	41 / M	484/06	+	8 mon	+	+	-	-	-	-	-	-	-	DU	+
50	Mahalingam	53 / M	496/06	+	10mon	-	-	-	-	-	-	-	-	-	DU	-

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Sl. No.	Name	Age / Sex	IP No.	Endoscopy	RUT
1	Saroja	24 / F	1402/05	N	-
2	Muthusamy	47 / M	1468/05	N	-
3	Ramesh	25 / M	1469/05	N	+
4	Rakkammal	49 / F	1507/05	N	-
5	Jothi	25 / F	1510/05	N	-
6	Manohar	36 / M	1529/05	N	-
7	Suresh	18 / M	1532/05	N	-
8	Lakshmi	46 / F	1606/05	N	-
9	Antony	34 / M	1686/05	N	+
10	Fathima	68 / F	1708/05	N	-
11	Kaliammal	39 / F	1744/05	N	-
12	Angammal	56 / F	1755/05	N	-
13	Subramani	42 / M	1794/05	N	-

Sl. No.	Name	Age / Sex	IP No.	Endoscopy	RUT
14	Cellammal	36 / F	1808/05	N	+
15	Mariappan	32 / M	1812/05	N	-
16	Thangamani	58 / F	1822/05	N	-
17	Selvi	29 / F	1840/05	N	-
18	Kannan	36 / M	1898/05	N	-
19	Mariyayee	32 / F	1906/05	N	+
20	Mani	24 / M	1914/05	N	-
21	Vasantha	46 / F	2010/05	N	-
22	Akbar	38 / M	2004/05	N	-
23	Chellammal	40 / F	2120/05	N	+
24	Dhanalakshmi	36 / F	2301/05	N	-
25	Rajeswari	49 / F	2309/05	N	+
26	Gokila	20 / F	2340/05	N	-
27	Shantha	35 / F	2344/05	N	-
28	Sangappan	64 / M	2564/05	N	+

Sl. No.	Name	Age / Sex	IP No.	Endoscopy	RUT
29	Mary	47 / F	2674/05	N	-
30	Devi	16 / F	2682/05	N	-
31	Veerammal	44 / F	2725/05	N	-
32	Ganesan	32 / M	2734/05	N	+
33	Gowri	33 / F	2844/05	N	-
34	Arumugam	28 / M	2938/05	N	-
35	Raman	38 / M	2961/05	N	+
36	Mohammed Ismail	71 / M	3055/05	N	-
37	Gunasamy	44 / M	3088/05	N	+
38	Umarani	22 / F	84/06	N	-
39	Jeevanandham	34 / M	123/06	N	-
40	Sivabackiam	53 / F	241/06	N	-
41	Subbulakshmi	46 / F	244/06	N	+
42	Hanifa	74 / M	268/06	N	-
43	Devi	26 / F	279/06	N	-

Sl. No.	Name	Age / Sex	IP No.	Endoscopy	RUT
44	Parvathy	55 / F	323/06	N	+
45	Lakshmi	43 / F	344/06	N	-
46	Radha	27 / F	361/06	N	-
47	Mani	42 / M	394/06	N	+
48	Kaliammal	44 / F	424/06	N	-
49	Ganesh	26 / M	453/06	N	-
50	Sundarammal	36 / F	464/06	N	-